(FILE 'HOME' ENTERED AT 09:22:05 ON 13 SEP 2003)

FILE 'MEDLINE, CAPLUS, EMBASE, BIOSIS' ENTERED AT 09:22:32 ON 13 SEP 2003

L1	373	S (BONZO OR STRL33 OR TYMSTR OR CXCR6 OR CXCL16)
L2	163	DUP REM L1 (210 DUPLICATES REMOVED)
L3	384571	S L2 AND CHEMOKINE OR HIV OR SIV
L4	151	S L2 AND (CHEMOKINE OR HIV OR SIV)
L5	46	S L4 AND PY<2000

=> d ibib abs 1-46 l5

L5 ANSWER 1 OF 46 MEDLINE on STN
ACCESSION NUMBER: 2000070533 MEDLINE
DOCUMENT NUMBER: 20070533 PubMed ID: 10602405
TITLE: **Chemokine** receptors and virus entry in the central nervous system.
AUTHOR: Gabuzda D; Wang J
CORPORATE SOURCE: Department of Cancer Immunology & AIDS, Dana-Farber Cancer Institute, JF712, 44 Binney Street, Boston, Massachusetts, MA 02115, USA.
CONTRACT NUMBER: AI 28691 (NIAID)
NS35734 (NINDS)
NS37277 (NINDS)
+
SOURCE: JOURNAL OF NEUROVIROLOGY, **(1999 Dec)** 5 (6) 643-58. Ref: 171
Journal code: 9508123. ISSN: 1355-0284.
PUB. COUNTRY: ENGLAND: United Kingdom
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
General Review; (REVIEW)
(REVIEW, TUTORIAL)
LANGUAGE: English
FILE SEGMENT: Priority Journals; AIDS
ENTRY MONTH: 200002
ENTRY DATE: Entered STN: 20000229
Last Updated on STN: 20000229
Entered Medline: 20000215

AB Several members of the **chemokine** receptor family are used as coreceptors together with CD4 for **HIV** and **SIV** entry in the central nervous system (CNS). CCR5 is the major coreceptor for **HIV**-1 infection of macrophages and microglia, the major target cells for **HIV**-1 infection in the CNS. CXCR4 and CCR3 are also expressed on microglia and can mediate infection by certain **HIV**-1 isolates but at lower efficiency than CCR5. Additional **chemokine** receptors that can function as **HIV**-1 and **SIV** coreceptors for a subset of viruses are expressed in the brain (i.e. Apj, CX3CR1, **STRL33/BONZO**, and gpr1), but their role in CNS infection has not been defined. The expression of CXCR4, and possibly other **chemokine** receptors, on subpopulations of neurons and glial cells may contribute to mechanisms of CNS injury that are independent of viral infection. Understanding the role of **chemokine** receptors and their **chemokine** ligands in **HIV**-1 and **SIV** infection of the CNS will elucidate mechanisms of viral tropism and pathogenesis and advance the development of new therapeutic strategies.

L5 ANSWER 2 OF 46 MEDLINE on STN
ACCESSION NUMBER: 2000032078 MEDLINE
DOCUMENT NUMBER: 20032078 PubMed ID: 10562499
TITLE: Nonproductive human immunodeficiency virus type 1 infection of human fetal astrocytes: independence from CD4 and major **chemokine** receptors.
AUTHOR: Sabri F; Tresoldi E; Di Stefano M; Polo S; Monaco M C; Verani A; Fiore J R; Lusso P; Major E; Chiodi F; Scarlatti G
CORPORATE SOURCE: Microbiology and Tumorbiology Center, Karolinska Institute, Doktorsringen 13, Stockholm, 17177, Sweden.
SOURCE: VIROLOGY, **(1999 Nov 25)** 264 (2) 370-84.
Journal code: 0110674. ISSN: 0042-6822.
PUB. COUNTRY: United States
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals; AIDS

OTHER SOURCE: GENBANK-AF194975; GENBANK-AF194976; GENBANK-AF194977; GENBANK-AF194978; GENBANK-AF194979; GENBANK-AF194980
ENTRY MONTH: 200001
ENTRY DATE: Entered STN: 20000114
Last Updated on STN: 20000114
Entered Medline: 20000104

AB Human immunodeficiency virus type 1 (**HIV**-1) infection of the brain is associated with neurological manifestations both in adults and in children. The primary target for **HIV**-1 infection in the brain is the microglia, but astrocytes can also be infected. We tested 26 primary **HIV**-1 isolates for their capacity to infect human fetal astrocytes in culture. Eight of these isolates, independent of their biological phenotype and **chemokine** receptor usage, were able to infect astrocytes. Although no sustained viral replication could be demonstrated, the virus was recovered by coculture with receptive cells such as macrophages or on stimulation with interleukin-1beta. To gain knowledge into the molecular events that regulate attachment and penetration of **HIV**-1 in astrocytes, we investigated the expression of several **chemokine** receptors. Fluorocytometry and calcium-mobilization assay did not provide evidence of expression of any of the major **HIV**-1 coreceptors, including CXCR4, CCR5, CCR3, and CCR2b, as well as the CD4 molecule on the cell surface of human fetal astrocytes. However, mRNA transcripts for CXCR4, CCR5, **Bonzo/STRL33/TYMSTR**, and APJ were detected by RT-PCR. Furthermore, infection of astrocytes by **HIV**-1 isolates with different **chemokine** receptor usage was not inhibited by the **chemokines** SDF-1beta, RANTES, MIP-1beta, or MCP-1 or by antibodies directed against the third variable region or the CD4 binding site of gp120. These data show that astrocytes can be infected by primary **HIV**-1 isolates via a mechanism independent of CD4 or major **chemokine** receptors. Furthermore, astrocytes are potential carriers of latent **HIV**-1 and on activation may be implicated in spreading the infection to other neighbouring cells, such as microglia or macrophages.

L5 ANSWER 3 OF 46 MEDLINE on STN
ACCESSION NUMBER: 1999391177 MEDLINE
DOCUMENT NUMBER: 99391177 PubMed ID: 10463536
TITLE: Blocking **HIV** co-receptors by **chemokines**
.
AUTHOR: Virelizier J L
CORPORATE SOURCE: Unite d'Immunologie Virale, Institut Pasteur, Paris, France.
SOURCE: DEVELOPMENTS IN BIOLOGICAL STANDARDIZATION, **(1999)** 97 105-9. Ref: 15
Journal code: 0427140. ISSN: 0301-5149.
PUB. COUNTRY: Switzerland
DOCUMENT TYPE: (LECTURES)
General Review; (REVIEW)
(REVIEW, TUTORIAL)
LANGUAGE: English
FILE SEGMENT: Priority Journals; AIDS
ENTRY MONTH: 199910
ENTRY DATE: Entered STN: 19991101
Last Updated on STN: 19991101
Entered Medline: 19991021

AB Specific **chemokines** can block **HIV** entry and replication because they antagonize the common strategy of lentiviruses to use **chemokine** receptors for infecting CD4+ cells of the body, especially lymphocytes and cells of the monocytic lineage. This raised intense academical and therapeutical interest. The antiviral potency of these **chemokines** is indeed remarkable, but depends on the **chemokine** and the **HIV** isolate used. This is because

HIV appears to use many co-receptors, alternatively or in addition to the CCR5 co-receptor. These include CCR3, CXCR4, **STRL33/ Bonzo/TYMSTR**, and BOB. The CC **chemokines** RANTES, MIP-1alpha, MIP-1beta, and Eotaxin can suppress the replication of CCR5- and CCR3-dependent viruses, while SDF-1 alpha/beta suppresses that of CXCR4-dependent strains. Although no general rule can be drawn at present, it appears that chronic **HIV** infection may give rise to viruses which, instead of using preferentially or exclusively CCR5, are capable of using more than one co-receptor. This underlines the need for assaying the tropism of primary isolates, using both fusion assays and protection of activated lymphocyte cultures by one or more antiviral **chemokines** or **chemokine** antagonists.

L5 ANSWER 4 OF 46 MEDLINE on STN
ACCESSION NUMBER: 1999329192 MEDLINE
DOCUMENT NUMBER: 99329192 PubMed ID: 10400765
TITLE: Patterns of **chemokine** receptor fusion cofactor utilization by human immunodeficiency virus type 1 variants from the lungs and blood.
AUTHOR: Singh A; Besson G; Mobasher A; Collman R G
CORPORATE SOURCE: Pulmonary and Critical Care Division, Department of Medicine, University of Pennsylvania School of Medicine, Philadelphia, Pennsylvania 19104-6060, USA.
CONTRACT NUMBER: HL 58004 (NHLBI)
SOURCE: JOURNAL OF VIROLOGY, **(1999 Aug)** 73 (8) 6680-90. Journal code: 0113724. ISSN: 0022-538X.
PUB. COUNTRY: United States
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals; AIDS
ENTRY MONTH: 199908
ENTRY DATE: Entered STN: 19990910
Last Updated on STN: 19990910
Entered Medline: 19990824

AB Human immunodeficiency virus type 1 (**HIV**-1) infection is highly compartmentalized, with distinct viral genotypes being found in the lungs, brain, and other organs compared with blood. CCR5 and CXCR4 are the principal **HIV**-1 coreceptors, and a number of other molecules support entry in vitro but their roles in vivo are uncertain. To address the relationship between tissue compartmentalization and the selective use of entry coreceptors, we generated functional env clones from primary isolates derived from the lungs and blood of three infected individuals and analyzed their use of the principal, secondary, orphan, and virus-encoded coreceptors for fusion. All Env proteins from lung viruses used CCR5 but not CXCR4, while those from blood viruses used CCR5 or CXCR4 or both. The orphan receptor APJ was widely used for fusion by Env proteins from both blood and lung viruses, but none used the cytomegalovirus-encoded receptor US28. Fusion mediated by the secondary coreceptors CCR2b, CCR3, CCR8, and CX3CR1 and orphan receptors GPR1, GPR15, and **STRL33** was variable and heterogeneous, with relatively broad utilization by env clones from isolates of one subject but limited use by env clones from the other two subjects. However, there was no clear distinction between blood and lung viruses in secondary or orphan coreceptor fusion patterns. In contrast to fusion, none of the secondary or orphan receptors enabled efficient productive infection. These results confirm, at the level of cofactor utilization, previous observations that **HIV**-1 populations in the lungs and blood are biologically distinct and demonstrate diversity within lung-derived as well as blood-derived quasispecies. However, the heterogeneity in coreceptor utilization among clones from each isolate and the lack of clear distinction between lung- and blood-derived Env proteins argue against selective coreceptor utilization as a major determinant of compartmentalization.

L5 ANSWER 5 OF 46 MEDLINE on STN
ACCESSION NUMBER: 1999292828 MEDLINE
DOCUMENT NUMBER: 99292828 PubMed ID: 10364284
TITLE: Effects of soluble CD4 on simian immunodeficiency virus infection of CD4-positive and CD4-negative cells.
AUTHOR: Schenten D; Marcon L; Karlsson G B; Parolin C; Kodama T; Gerard N; Sodroski J
CORPORATE SOURCE: Department of Cancer Immunology and AIDS, Dana-Farber Cancer Institute, Harvard Medical School, Boston, Massachusetts 02115, USA.
CONTRACT NUMBER: AI24755 (NIAID)
AI28691 (NIAID)
AI41851 (NIAID)
SOURCE: JOURNAL OF VIROLOGY, **(1999 Jul)** 73 (7) 5373-80.
Journal code: 0113724. ISSN: 0022-538X.
PUB. COUNTRY: United States
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals; AIDS
ENTRY MONTH: 199907
ENTRY DATE: Entered STN: 19990806
Last Updated on STN: 19990806
Entered Medline: 19990723

AB A soluble form of the CD4 receptor (sCD4) can either enhance or inhibit the infection of cells by simian immunodeficiency virus (**SIV**) and human immunodeficiency virus. We investigated the basis for these varying effects by studying the entry of three **SIV** isolates into CD4-positive and CD4-negative cells expressing different **chemokine** receptors. Infection of CD4-negative cells depended upon the viral envelope glycoproteins and upon the **chemokine** receptor, with CCR5 and gpr15 being more efficient than **STRL33**. Likewise, enhancement of infection by sCD4 was observed when CCR5- and gpr15-expressing target cells were used but not when those expressing **STRL33** were used. The sCD4-mediated enhancement of virus infection of CD4-negative, CCR5-positive cells was related to the sCD4-induced increase in binding of the viral gp120 envelope glycoprotein to CCR5. Inhibitory effects of sCD4 could largely be explained by competition for virus attachment to cellular CD4 rather than other detrimental effects on virus infectivity (e.g., disruption of the envelope glycoprotein spike). Consistent with this, the sCD4-activated **SIV** envelope glycoprotein intermediate on the virus was long-lived. Thus, the net effect of sCD4 on **SIV** infectivity appears to depend upon the degree of enhancement of **chemokine** receptor binding and upon the efficiency of competition for cellular CD4.

L5 ANSWER 6 OF 46 MEDLINE on STN
ACCESSION NUMBER: 1999281902 MEDLINE
DOCUMENT NUMBER: 99281902 PubMed ID: 10355771
TITLE: Coreceptor usage of BOB/GPR15 and **Bonzo/ STRL33** by primary isolates of human immunodeficiency virus type 1.
AUTHOR: Pohlmann S; Krumbiegel M; Kirchhoff F
CORPORATE SOURCE: Institute for Clinical and Molecular Virology, University of Erlangen-Nurnberg, Erlangen, Germany.
SOURCE: JOURNAL OF GENERAL VIROLOGY, **(1999 May)** 80 (Pt 5) 1241-51.
Journal code: 0077340. ISSN: 0022-1317.
PUB. COUNTRY: ENGLAND: United Kingdom
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals; AIDS
ENTRY MONTH: 199906
ENTRY DATE: Entered STN: 19990628
Last Updated on STN: 20000303

Entered Medline: 19990615

AB Primary isolates of human and simian immunodeficiency viruses (**HIV** and **SIV**) use the **chemokine** receptor CCR5, in association with CD4, as coreceptor. During AIDS progression, **HIV** -1 and **HIV**-2 often adapt to use additional cofactors, particularly CXCR4. In contrast, **SIV** isolates do not use CXCR4, but other coreceptors such as BOB/GPR15 and **Bonzo/STRL33** . Only limited information is currently available on usage of BOB/GPR15 and **Bonzo/STRL33** by **HIV**-1. Therefore, we investigated a panel of gp160 clones from 15 primary isolates, representing 5 different subtypes, for utilization of these cofactors. The majority of **HIV**-1 envelopes mediated entry into BOB/GPR15-expressing cells, albeit often with low efficiency. Usage of **Bonzo/STRL33** was less common and usually inefficient. To investigate if **HIV**-1 entry via these orphan receptors is sufficient to allow virus replication, 15 uncloned primary **HIV**-1 isolates and 7 molecular clones were used to infect target cells expressing CD4 and **Bonzo/STRL33** or BOB/GPR15. Three primary isolates and two molecular clones replicated efficiently in cells expressing BOB/GPR15. Two of these isolates were X4-tropic, two were R5X4-tropic and one was R5-tropic. In contrast, none of the **HIV** -1 variants showed significant levels of replication in **Bonzo/ STRL33**-expressing cells. Our data show that some **HIV**-1 isolates of different genetic subtype and of different biological phenotype use BOB/GPR15 for productive infection and suggest that this cofactor may play a role in **HIV**-1 pathogenesis and transmission.

L5 ANSWER 7 OF 46 MEDLINE on STN

ACCESSION NUMBER: 1999260286 MEDLINE
DOCUMENT NUMBER: 99260286 PubMed ID: 10331443
TITLE: Phenotypic characteristics of human immunodeficiency virus type 1 subtype C isolates of Ethiopian AIDS patients.
AUTHOR: Bjorndal A; Sonnerborg A; Tscherning C; Albert J; Fenyo E M
CORPORATE SOURCE: Microbiology and Tumor Biology Center, Karolinska Institute, Stockholm, Sweden.. Asa.Bjorndal@mtc.ki.se
SOURCE: AIDS RESEARCH AND HUMAN RETROVIRUSES, **(1999 May 1)** 15 (7) 647-53.
Journal code: 8709376. ISSN: 0889-2229.
PUB. COUNTRY: United States
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals; AIDS
ENTRY MONTH: 199908
ENTRY DATE: Entered STN: 19990820
Last Updated on STN: 19990820
Entered Medline: 19990806

AB It has been estimated that, to date, about 48% of all **HIV** -infected people in the world carry **HIV**-1 subtype C virus. Therefore, it is of great importance to gain better knowledge about the genetic and biological characteristics of this virus subtype. In the present study, the biological properties of **HIV**-1 isolates obtained from nine Ethiopian patients with AIDS were studied. DNA sequencing of the V3 loop of gp120 classified the isolates as subtype C. In primary isolation cultures, virus infection was accompanied by syncytium formation and cell lysis. Interestingly, when examining the growth in primary monocyte-macrophage cultures, initial low-level virus replication was followed by a nonproductive state, from which virus could be rescued by cocultivation with Jurkat(tat) cells. Furthermore, none of the isolates replicated in T cell lines (CEM, MT-2, HuT-78, and H9) or in the promonocytic cell line U937 clone 2. All isolates could use CCR5 as coreceptor, whereas no isolates could use CCR2b, CCR3, CCR5, CXCR4, **Bonzo/STRL33**, or BOB/GPR15. The genotype of the V3 region correlated with the MT-2 negative/non-syncytium-inducing (NSI) phenotype. Comparative studies revealed that the scarcity of CXCR4 usage

as well as other phenotypic characteristics of subtype C isolates distinguish this subtype. On the basis of these data, we suggest that in addition, factors other than viral phenotype may govern the pathogenic potential of subtype C isolates.

L5 ANSWER 8 OF 46 MEDLINE on STN
ACCESSION NUMBER: 1999174047 MEDLINE
DOCUMENT NUMBER: 99174047 PubMed ID: 10074200
TITLE: Will multiple coreceptors need to be targeted by inhibitors of human immunodeficiency virus type 1 entry?.
AUTHOR: Zhang Y J; Moore J P
CORPORATE SOURCE: Aaron Diamond AIDS Research Center, The Rockefeller University, New York, New York, USA.
CONTRACT NUMBER: AI41420 (NIAID)
SOURCE: JOURNAL OF VIROLOGY, **(1999 Apr)** 73 (4) 3443-8.
Journal code: 0113724. ISSN: 0022-538X.
PUB. COUNTRY: United States
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals; AIDS
ENTRY MONTH: 199905
ENTRY DATE: Entered STN: 19990517
Last Updated on STN: 19990517
Entered Medline: 19990506

AB Despite being able to use the **Bonzo** coreceptor as efficiently as CCR5 in transfected cells, pediatric human immunodeficiency virus type 1 isolate P6 was unable to replicate in peripheral blood mononuclear cells (PBMC) lacking the CCR5 receptor. Furthermore, its replication in wild-type PBMC was completely inhibited by inhibitors of CCR5-mediated entry. Similarly, maternal isolate M6 could use CCR5, CXCR4, **Bonzo**, and other coreceptors in transfected cells but was completely sensitive to inhibitors of CCR5- and CXCR4-mediated entry when grown in PBMC. The ability of these viruses to use coreceptors in addition to CCR5 and CXCR4 in vitro was, therefore, irrelevant to their drug sensitivity in primary cells. We argue that CCR5 and CXCR4 should remain the primary targets for antiviral drug development, pending strong evidence to the contrary.

L5 ANSWER 9 OF 46 MEDLINE on STN
ACCESSION NUMBER: 1999139018 MEDLINE
DOCUMENT NUMBER: 99139018 PubMed ID: 9971818
TITLE: V3 recombinants indicate a central role for CCR5 as a coreceptor in tissue infection by human immunodeficiency virus type 1.
AUTHOR: Chan S Y; Speck R F; Power C; Gaffen S L; Chesebro B; Goldsmith M A
CORPORATE SOURCE: Gladstone Institute of Virology and Immunology, San Francisco, California, USA.
SOURCE: JOURNAL OF VIROLOGY, **(1999 Mar)** 73 (3) 2350-8.
Journal code: 0113724. ISSN: 0022-538X.
PUB. COUNTRY: United States
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals; AIDS
ENTRY MONTH: 199903
ENTRY DATE: Entered STN: 19990316
Last Updated on STN: 19990316
Entered Medline: 19990304

AB Binding of the human immunodeficiency virus type 1 (**HIV**-1) envelope glycoprotein gp120 to both CD4 and one of several **chemokine** receptors (coreceptors) permits entry of virus into target cells. Infection of tissues may establish latent viral reservoirs as well as cause direct pathologic effects that manifest as clinical disease such as **HIV**-associated dementia. We sought to identify

the critical coreceptors recognized by **HIV**-1 tissue-derived strains as well as to correlate these coreceptor preferences with site of infection and dementia diagnosis. To reconstitute coreceptor use, we cloned **HIV**-1 envelope V3 sequences encoding the primary determinants of coreceptor specificity from 13 brain-derived and 6 colon-derived viruses into an isogenic (NL4-3) viral background. All V3 recombinants utilized the **chemokine** receptor CCR5 uniformly and efficiently as a coreceptor but not CXCR4, BOB/GPR15, or **Bonzo/ STRL33**. Other receptors such as CCR3, CCR8, and US28 were inefficiently and variably used as coreceptors by various envelopes. CCR5 without CD4 present did not allow for detectable infection by any of the tested recombinants. In contrast to the pathogenic switch in coreceptor specificity frequently observed in comparisons of blood-derived viruses early after **HIV**-1 seroconversion and after onset of AIDS, the characteristics of these V3 recombinants suggest that CCR5 is a primary coreceptor for brain- and colon-derived viruses regardless of tissue source or diagnosis of dementia. Therefore, tissue infection may not depend significantly on viral envelope quasispeciation to broaden coreceptor range but rather selects for CCR5 use throughout disease progression.

L5 ANSWER 10 OF 46 MEDLINE on STN
ACCESSION NUMBER: 1999139017 MEDLINE
DOCUMENT NUMBER: 99139017 PubMed ID: 9971817
TITLE: Primary human immunodeficiency virus type 2 (**HIV** -2) isolates, like **HIV**-1 isolates, frequently use CCR5 but show promiscuity in coreceptor usage.
AUTHOR: Morner A; Bjorndal A; Albert J; Kewalramani V N; Littman D R; Inoue R; Thorstensson R; Fenyo E M; Bjorling E
CORPORATE SOURCE: Microbiology and Tumorbiology Center (MTC), Karolinska Institute, Swedish Institute for Infectious Disease Control, Stockholm, Sweden.. andreas.morner@mtc.ki.se
SOURCE: JOURNAL OF VIROLOGY, **(1999 Mar)** 73 (3) 2343-9. Journal code: 0113724. ISSN: 0022-538X.
PUB. COUNTRY: United States
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals; AIDS
ENTRY MONTH: 199903
ENTRY DATE: Entered STN: 19990316
Last Updated on STN: 19990316
Entered Medline: 19990304

AB Coreceptor usage of primary human immunodeficiency virus type 1 (**HIV**-1) isolates varies according to biological phenotype. The **chemokine** receptors CCR5 and CXCR4 are the major coreceptors that, together with CD4, govern **HIV**-1 entry into cells. Since CXCR4 usage determines the biological phenotype for **HIV**-1 isolates and is more frequent in patients with immunodeficiency, it may serve as a marker for viral virulence. This possibility prompted us to study coreceptor usage by **HIV**-2, known to be less pathogenic than **HIV**-1. We tested 11 primary **HIV**-2 isolates for coreceptor usage in human cell lines: U87 glioma cells, stably expressing CD4 and the **chemokine** receptor CCR1, CCR2b, CCR3, CCR5, or CXCR4, and GHOST(3) osteosarcoma cells, coexpressing CD4 and CCR5, CXCR4, or the orphan receptor **Bonzo** or BOB. The indicator cells were infected by cocultivation with virus-producing peripheral blood mononuclear cells and by cell-free virus. Our results show that 10 of 11 **HIV**-2 isolates were able to efficiently use CCR5. In contrast, only two isolates, both from patients with advanced disease, used CXCR4 efficiently. These two isolates also promptly induced syncytia in MT-2 cells, a pattern described for **HIV**-1 isolates that use CXCR4. Unlike **HIV**-1, many of the **HIV**-2 isolates were promiscuous in their coreceptor usage in that they were able to use, apart from CCR5, one or more of the CCR1, CCR2b, CCR3, and BOB coreceptors.

Another difference between **HIV**-1 and **HIV**-2 was that the ability to replicate in MT-2 cells appeared to be a general property of **HIV**-2 isolates. Based on BOB mRNA expression in MT-2 cells and the ability of our panel of **HIV**-2 isolates to use BOB, we suggest that **HIV**-2 can use BOB when entering MT-2 cells. The results indicate no obvious link between viral virulence and the ability to use a multitude of coreceptors.

L5 ANSWER 11 OF 46 MEDLINE on STN
ACCESSION NUMBER: 1999099061 MEDLINE
DOCUMENT NUMBER: 99099061 PubMed ID: 9882375
TITLE: Coreceptor specificity of temporal variants of simian immunodeficiency virus Mne.
AUTHOR: Kimata J T; Gosink J J; KewalRamani V N; Rudensey L M; Littman D R; Overbaugh J
CORPORATE SOURCE: Department of Microbiology, University of Washington, Seattle, Washington 98195, USA.
CONTRACT NUMBER: AI34251 (NIAID)
F32 AI09337 (NIAID)
T32 CA09229 (NCI)
SOURCE: JOURNAL OF VIROLOGY, **(1999 Feb)** 73 (2) 1655-60.
Journal code: 0113724. ISSN: 0022-538X.
PUB. COUNTRY: United States
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals; AIDS
ENTRY MONTH: 199902
ENTRY DATE: Entered STN: 19990301
Last Updated on STN: 19990301
Entered Medline: 19990218
AB The simian immunodeficiency virus (**SIV**) Mne envelope undergoes genetic changes that alter tropism, syncytium-inducing capacity, and antigenic properties of the emerging variant virus population during the course of an infection. Here we investigated whether the mutations in envelope of SIVMne also influence coreceptor usage. The data demonstrate that the infecting macrophage-tropic SIVMne clone as well as the envelope variants that are selected during the course of disease progression all recognize both CCR5 and Bob (GPR15) but not **Bonzo** (**STRL33**), CXCR4, or CCR3. Although it remains to be determined if there are other coreceptors specific for dualtropic or T-cell-tropic variants of SIVMne that emerge during late stages of infection, these data suggest that such **SIV** variants that evolve in pathogenic infections do not lose the ability to recognize CCR5 or Bob/GPR15.

L5 ANSWER 12 OF 46 MEDLINE on STN
ACCESSION NUMBER: 1999009390 MEDLINE
DOCUMENT NUMBER: 99009390 PubMed ID: 9791028
TITLE: Use of GPR1, GPR15, and **STRL33** as coreceptors by diverse human immunodeficiency virus type 1 and simian immunodeficiency virus envelope proteins.
AUTHOR: Edinger A L; Hoffman T L; Sharron M; Lee B; O'Dowd B; Doms R W
CORPORATE SOURCE: Department of Pathology and Laboratory Medicine, University of Pennsylvania, 34th Street and Civic Center Boulevard, Philadelphia, Pennsylvania, 19104, USA.
CONTRACT NUMBER: 2T32GM07170 (NIGMS)
R01-AI40880 (NIAID)
SOURCE: VIROLOGY, **(1998 Sep 30)** 249 (2) 367-78.
Journal code: 0110674. ISSN: 0042-6822.
PUB. COUNTRY: United States
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals; AIDS
ENTRY MONTH: 199811

ENTRY DATE: Entered STN: 19990106
Last Updated on STN: 19990106
Entered Medline: 19981110

AB Human and simian immunodeficiency viruses (**HIV** and **SIV**, respectively) use **chemokine** receptors as coreceptors along with CD4 to mediate viral entry. Several orphan receptors, including GPR1, GPR15, and **STRL33**, can also serve as coreceptors for a more limited number of **HIV** and **SIV** isolates. We investigated whether these orphan receptors could function as efficient coreceptors for a diverse group of **HIV** and **SIV** envelopes (Envs) in comparison with the principal coreceptors CCR5 and CXCR4. We found that a limited number of **HIV**-1 isolates could mediate inefficient cell-cell fusion with the orphan receptors relative to CCR5 and CXCR4; however, none of the orphan receptors tested could support pseudotype virus infection despite robust infection via CCR5 or CXCR4. All except one of the **SIV** Envs tested mediated some degree of cell-cell fusion and pseudotype infection, with target cells expressing at least one of these orphan receptors, although CCR5 proved to be the most efficient coreceptor for infection. Only one **SIV** Env protein, BK28, could mediate infection using GPR1 as a coreceptor, albeit much less efficiently than with CCR5. In addition, use of these coreceptors did not correlate with the published tropism of the **SIV** clones and was strictly CD4 dependent for both **SIV** and **HIV**. We also examined the expression of these molecules in cell lines and primary cells widely used for virus propagation and as targets for infection. All cells examined expressed **STRL33**, a more limited number expressed GPR15, and GPR1 was much more restricted in its expression pattern. Taken together, our results indicate that GPR15 and **STRL33** are rarely used by **HIV**-1 but are more frequently used by **SIV** strains, although not in a manner that correlates with **SIV** tropism.

L5 ANSWER 13 OF 46 MEDLINE on STN
ACCESSION NUMBER: 1998440604 MEDLINE
DOCUMENT NUMBER: 98440604 PubMed ID: 9765485
TITLE: Use of coreceptors other than CCR5 by non-syncytium-inducing adult and pediatric isolates of human immunodeficiency virus type 1 is rare in vitro.
AUTHOR: Zhang Y J; Dragic T; Cao Y; Kostrikis L; Kwon D S; Littman D R; KewalRamani V N; Moore J P
CORPORATE SOURCE: Aaron Diamond AIDS Research Center, The Rockefeller University, New York University School of Medicine, New York, New York 10016, USA.
CONTRACT NUMBER: AI41420 (NIAID)
SOURCE: JOURNAL OF VIROLOGY, **(1998 Nov)** 72 (11) 9337-44.
Journal code: 0113724. ISSN: 0022-538X.
PUB. COUNTRY: United States
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals; AIDS
ENTRY MONTH: 199811
ENTRY DATE: Entered STN: 19990106
Last Updated on STN: 19990106
Entered Medline: 19981105

AB We have tested a panel of pediatric and adult human immunodeficiency virus type 1 (**HIV**-1) primary isolates for the ability to employ the following proteins as coreceptors during viral entry: CCR1, CCR2b, CCR3, CCR4, CCR5, CCR8, CXCR4, **Bonzo**, BOB, GPR1, V28, US28, and APJ. Most non-syncytium-inducing isolates could utilize only CCR5. All syncytium-inducing viruses used CXCR4, some also employed V28, and one (DH123) used CCR8 and APJ as well. A longitudinal series of **HIV**-1 subtype B isolates from an infected infant and its mother utilized **Bonzo** efficiently, as well as CCR5. The maternal isolates, which

were syncytium inducing, also used CXCR4, CCR8, V28, and APJ.

L5 ANSWER 14 OF 46 MEDLINE on STN
ACCESSION NUMBER: 1998440599 MEDLINE
DOCUMENT NUMBER: 98440599 PubMed ID: 9765480
TITLE: **Chemokine** coreceptor usage by diverse primary isolates of human immunodeficiency virus type 1.
AUTHOR: Zhang L; He T; Huang Y; Chen Z; Guo Y; Wu S; Kunstman K J; Brown R C; Phair J P; Neumann A U; Ho D D; Wolinsky S M
CORPORATE SOURCE: The Aaron Diamond AIDS Research Center, The Rockefeller University, New York, New York 10016, USA.. izhang@adarc.org
CONTRACT NUMBER: AI-35168 (NIAID)
AI24518 (NIAID)
HD-31756 (NICHD)
+
SOURCE: JOURNAL OF VIROLOGY, **(1998 Nov)** 72 (11) 9307-12.
Journal code: 0113724. ISSN: 0022-538X.
PUB. COUNTRY: United States
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals; AIDS
ENTRY MONTH: 199811
ENTRY DATE: Entered STN: 19990106
Last Updated on STN: 19990106
Entered Medline: 19981105

AB We tested **chemokine** receptor subset usage by diverse, well-characterized primary viruses isolated from peripheral blood by monitoring viral replication with CCR1, CCR2b, CCR3, CCR5, and CXCR4 U87MG.CD4 transformed cell lines and **STRL33/BONZO/ TYMSTR** and GPR15/BOB HOS.CD4 transformed cell lines. Primary viruses were isolated from 79 men with confirmed human immunodeficiency virus type 1 (**HIV**-1) infection from the Chicago component of the Multicenter AIDS Cohort Study at interval time points. Thirty-five additional well-characterized primary viruses representing **HIV**-1 group M subtypes A, B, C, D, and E and group O and three primary simian immunodeficiency virus (**SIV**) isolates were also used for these studies. The restricted use of the CCR5 **chemokine** receptor for viral entry was associated with infection by a virus having a non-syncytium-inducing phenotype and correlated with a reduced rate of disease progression and a prolonged disease-free interval. Conversely, broadening **chemokine** receptor usage from CCR5 to both CCR5 and CXCR4 was associated with infection by a virus having a syncytium-inducing phenotype and correlated with a faster rate of CD4 T-cell decline and progression of disease. We also observed a greater tendency for infection with a virus having a syncytium-inducing phenotype in men heterozygous for the defective CCR5 Delta32 allele (25%) than in those men homozygous for the wild-type CCR5 allele (6%) (P = 0.03). The propensity for infection with a virus having a syncytium-inducing phenotype provides a partial explanation for the rapid disease progression among some men heterozygous for the defective CCR5 Delta32 allele. Furthermore, we did not identify any primary viruses that used CCR3 as an entry cofactor, despite this CC **chemokine** receptor being expressed on the cell surface at a level commensurate with or higher than that observed for primary peripheral blood mononuclear cells. Whereas isolates of primary viruses of **SIV** also used **STRL33/BONZO/TYMSTR** and GPR15/BOB, no primary isolates of **HIV**-1 used these particular **chemokine** receptor-like orphan molecules as entry cofactors, suggesting a limited contribution of these other **chemokine** receptors to viral evolution. Thus, despite the number of **chemokine** receptors implicated in viral entry, CCR5 and CXCR4 are likely to be the physiologically relevant **chemokine** receptors used as entry cofactors in vivo by diverse strains of primary viruses isolated from blood.

L5 ANSWER 15 OF 46 MEDLINE on STN
ACCESSION NUMBER: 1998435802 MEDLINE
DOCUMENT NUMBER: 98435802 PubMed ID: 9764773
TITLE: Adaptation to promiscuous usage of CC and CXC-**chemokine** coreceptors in vivo correlates with **HIV**-1 disease progression.
AUTHOR: Xiao L; Rudolph D L; Owen S M; Spira T J; Lal R B
CORPORATE SOURCE: HIV/Retrovirus Diseases Branch, Division of AIDS, STD, and TB Laboratory Research, National Center for Infectious Diseases, Centers for Disease Control and Prevention, Public Health Services, US Department of Health and Human Serv.
SOURCE: AIDS, **(1998 Sep 10)** 12 (13) F137-43.
Journal code: 8710219. ISSN: 0269-9370.
PUB. COUNTRY: United States
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals; AIDS
ENTRY MONTH: 199812
ENTRY DATE: Entered STN: 19990115
Last Updated on STN: 20000303
Entered Medline: 19981218

AB OBJECTIVE: To study coreceptor usage of sequential primary **HIV**-1 isolates in a longitudinal follow-up cohort of **HIV**-1-infected men to understand its contribution to pathogenesis of **HIV** disease. DESIGN: Viral coreceptor usage of sequential primary isolates from **HIV**-1-infected individuals was examined at various timepoints and data was compared with CD4 cell counts, rates of disease progression and beta-**chemokine** production. METHODS: Fifty-eight sequential primary isolates were obtained from four rapid progressors, six late progressors, and three long-term nonprogressors (LTNP) and their coreceptor usage was examined by infection of peripheral blood mononuclear cells (PBMC) from donors with wild-type or non-functional CC-**chemokine** receptor (CCR)-5, and by infection of GHOST4 cells expressing CD4 and various **chemokine** receptors [CCR-1-CCR-5, CXC-**chemokine** receptor (CXCR)-4, BOB/GPR15, **BONZO/STRL33**]. Production of RANTES and macrophage inflammatory protein (MIP)-1beta was examined using unstimulated or phytohemagglutinin (PHA)-stimulated PBMC isolated from these individuals at multiple timepoints during infection. RESULTS: A switch from single CCR-5 coreceptor usage to multiple coreceptor usage occurred in all four rapid progressors and three out of six late progressors. In addition to the commonly used coreceptors CXCR-4, CCR-5, and CCR-3, some of the viruses isolated from patients in the terminal stage of infection also used CCR-1, CCR-2b, CCR-4, and BOB as coreceptors. The emergence of viral variants capable of utilizing multiple coreceptors generally preceded CD4 cell decline to < 200 x 10(6)/l and correlated with the onset of AIDS. In contrast, three LTNP maintained exclusive usage of CCR-5 over a period of 7-12 years post-infection. Endogenous production of RANTES and MIP-1beta by PBMC from LTNP was not significantly different from rapid and late progressors. However, PHA-driven production of both **chemokines** was significantly higher in LTNP, suggesting that in vivo activating stimuli might curtail **HIV** replication by inducing these **chemokines**. CONCLUSIONS: Viral variants capable of utilizing a broad range of coreceptors correlated with **HIV**-1 disease progression. In contrast, LTNP maintain exclusive usage of CCR-5 and produce higher levels of beta-**chemokines**. Thus, both viral and host determinants leading to the emergence of viral variants capable of using an expanded range of coreceptors may be likely determinants of disease progression.

L5 ANSWER 16 OF 46 MEDLINE on STN
ACCESSION NUMBER: 1998414254 MEDLINE

DOCUMENT NUMBER: 98414254 PubMed ID: 9743322
TITLE: Enhanced anti-**HIV**-1 activity and altered chemotactic potency of NH2-terminally processed macrophage-derived **chemokine** (MDC) imply an additional MDC receptor.
AUTHOR: Struyf S; Proost P; Sozzani S; Mantovani A; Wuyts A; De Clercq E; Schols D; Van Damme J
CORPORATE SOURCE: Laboratory of Molecular Immunology, Rega Institute for Medical Research, University of Leuven, Belgium.
SOURCE: JOURNAL OF IMMUNOLOGY, **(1998 Sep 15)** 161 (6) 2672-5.
Journal code: 2985117R. ISSN: 0022-1767.
PUB. COUNTRY: United States
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Abridged Index Medicus Journals; Priority Journals; AIDS
ENTRY MONTH: 199810
ENTRY DATE: Entered STN: 19981020
Last Updated on STN: 19981020
Entered Medline: 19981006

AB Posttranslational processing of **chemokines** increases (IL-8) or decreases (monocyte chemotactic protein-1) their chemotactic potency. Macrophage-derived **chemokine** (MDC) attracts monocytes, dendritic cells, activated lymphocytes, and NK cells and has reportedly anti-**HIV**-1 activity. Here we report that truncation of MDC by deletion of two NH2-terminal residues resulted in impaired binding to CC **chemokine** receptor (CCR)4, the only identified MDC receptor so far. Truncated MDC(3-69) failed to desensitize calcium mobilization by MDC(1-69) or thymus- and activation-regulated **chemokine** (TARC), another CCR4 ligand. MDC(3-69) lacked HUT-78 T cell chemotactic activity but retained its capacity to attract monocytes and to desensitize chemotaxis. Compared with MDC(1-69), MDC(3-69) had weak but enhanced antiviral activity against M- and T-tropic **HIV**-1 strains. Furthermore, both MDC forms failed to signal through the orphan receptors **Bonzo/STRL33** and BOB/GPR15 and to desensitize RANTES and stromal cell-derived factor (SDF)-1 responses in CCR5-transfected and CXC **chemokine** receptor (CXCR)4-transfected cells, respectively. These findings suggest that MDC recognizes another, yet unidentified, receptor. We conclude that minimal NH2-terminal truncation of MDC differentially affects its various immunologic functions.

L5 ANSWER 17 OF 46 MEDLINE on STN
ACCESSION NUMBER: 1998409662 MEDLINE
DOCUMENT NUMBER: 98409662 PubMed ID: 9736741
TITLE: **HIV** type I envelope determinants for use of the CCR2b, CCR3, **STRL33**, and APJ coreceptors.
AUTHOR: Hoffman T L; Stephens E B; Narayan O; Doms R W
CORPORATE SOURCE: Department of Pathology and Laboratory Medicine, University of Pennsylvania, Philadelphia, PA 19104, USA.
CONTRACT NUMBER: AI38492 (NIAID)
D49516
R01 AI-40880 (NIAID)
+
SOURCE: PROCEEDINGS OF THE NATIONAL ACADEMY OF SCIENCES OF THE UNITED STATES OF AMERICA, **(1998 Sep 15)** 95 (19) 11360-5.
Journal code: 7505876. ISSN: 0027-8424.
PUB. COUNTRY: United States
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals; AIDS
ENTRY MONTH: 199810
ENTRY DATE: Entered STN: 19990106
Last Updated on STN: 19990106

Entered Medline: 19981026

AB The envelope (Env) proteins of primate lentiviruses interact sequentially with CD4 and a coreceptor to infect cells. Changes in coreceptor use strongly influence viral tropism and pathogenesis. We followed the evolution of coreceptor use in pig-tailed macaques that developed severe CD4 T-cell loss during the derivation of a pathogenic simian **HIV** (SHIV) that contained the tat, rev, vpu, and env genes of the HXBc2 strain of **HIV**-1 in a genetic background of SIVmac239. The Env from the parental virus as well as one derived from the first macaque to develop AIDS exclusively used CXCR4 as a coreceptor, indicating that CXCR4 can function as a coreceptor in macaques even though it is rarely used by simian immunodeficiency viruses. One Env (Pnb5), obtained from a macrophage-tropic virus isolated from the cerebral spinal fluid, did not use CCR5 or CXCR4. Instead, it used CCR2b and to a lesser extent CCR3, **STRL33**, and APJ to infect cells. Chimeras between Pnb5 and the parental X4 Env indicated that the V3 loop is the major determinant of CXCR4 use, with other regions of Env influencing the efficiency with which this coreceptor was used. In contrast, the Pnb5 V1/2 and V3 regions in combination were both necessary and sufficient to confer full use of CCR2b, CCR3, **STRL33**, and APJ to the parental X4 Env protein. These results are consistent with a single, conserved binding site in Env that interacts with multiple coreceptors in conjunction with the V1/2 and V3 loops, and suggest that the V1/2 region plays a more important role in governing the use of CCR2b, CCR3, **STRL33**, and APJ than for CXCR4.

L5 ANSWER 18 OF 46 MEDLINE on STN
ACCESSION NUMBER: 1998406265 MEDLINE
DOCUMENT NUMBER: 98406265 PubMed ID: 9733901
TITLE: CXCR4 as a functional coreceptor for human immunodeficiency virus type 1 infection of primary macrophages.
AUTHOR: Simmons G; Reeves J D; McKnight A; Dejucq N; Hibbitts S; Power C A; Aarons E; Schols D; De Clercq E; Proudfoot A E; Clapham P R
CORPORATE SOURCE: Section of Virology, Chester Beatty Laboratories, Institute of Cancer Research, London SW3 6JB, United Kingdom.
SOURCE: JOURNAL OF VIROLOGY, **(1998 Oct)** 72 (10) 8453-7. Journal code: 0113724. ISSN: 0022-538X.
PUB. COUNTRY: United States
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals; AIDS
ENTRY MONTH: 199810
ENTRY DATE: Entered STN: 19981020
Last Updated on STN: 19981020
Entered Medline: 19981007

AB The coreceptors used by primary syncytium-inducing (SI) human immunodeficiency virus type 1 isolates for infection of primary macrophages were investigated. SI strains using only CXCR4 replicated equally well in macrophages with or without CCR5 and were inhibited by several different ligands for CXCR4 including SDF-1 and bicyclam derivative AMD3100. SI strains that used a broad range of coreceptors including CCR3, CCR5, CCR8, CXCR4, and **BONZO** infected CCR5-deficient macrophages about 10-fold less efficiently than CCR5(+) macrophages. Moreover, AMD3100 blocked infection of CCR5-negative macrophages by these strains. Our results therefore demonstrate that CXCR4, as well as CCR5, is used for infection of primary macrophages but provide no evidence for the use of alternative coreceptors.

L5 ANSWER 19 OF 46 MEDLINE on STN
ACCESSION NUMBER: 1998350791 MEDLINE
DOCUMENT NUMBER: 98350791 PubMed ID: 9686174
TITLE: **Chemokines**, lymphocytes, and **HIV**.
AUTHOR: Farber J M

CORPORATE SOURCE: Laboratory of Clinical Investigation, National Institute of Allergy and Infectious Diseases, Bethesda, MD 20892-1888, USA.. joshua_farber@nih.gov
SOURCE: BRAZILIAN JOURNAL OF MEDICAL AND BIOLOGICAL RESEARCH, **(1998 Jan)** 31 (1) 11-7. Ref: 40
Journal code: 8112917. ISSN: 0100-879X.
PUB. COUNTRY: Brazil
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
General Review; (REVIEW)
(REVIEW, TUTORIAL)
LANGUAGE: English
FILE SEGMENT: Priority Journals; AIDS
ENTRY MONTH: 199809
ENTRY DATE: Entered STN: 19980917
Last Updated on STN: 19980917
Entered Medline: 19980908

AB **Chemokines** are members of a family of more than 30 human cytokines whose best-described activities are as chemotactic factors for leukocytes and that are presumed to be important in leukocyte recruitment and trafficking. While many **chemokines** can act on lymphocytes, the roles of **chemokines** and their receptors in lymphocyte biology are poorly understood. The recent discoveries that **chemokines** can suppress infection by **HIV**-1 and that **chemokine** receptors serve, along with CD4, as obligate co-receptors for **HIV**-1 entry have lent urgency to studies on the relationships between **chemokines** and lymphocytes. My laboratory has characterized Mig and Crg-2/IP-10, **chemokines** that are induced by IFN-gamma and that specifically target lymphocytes, particularly activated T cells. We have demonstrated that the genes for these **chemokines** are widely expressed during experimental infections in mice with protozoan and viral pathogens, but that the patterns of mig and crg-2 expression differed, suggesting non-redundant roles in vivo. Our related studies to identify new **chemokine** receptors from activated lymphocytes resulted in the cloning of STRL22 and **STRL33**. We and others have shown that STRL22 is a receptor for the CC **chemokine** MIP-3 alpha, and STRL22 has been renamed CCR6. Although **STRL33** remains an orphan receptor, we have shown that it can function as a co-receptor for **HIV**-1 envelope glycoproteins, and that it is active with a broader range of **HIV** -1 envelope glycoproteins than the major co-receptors described to date. The ability of **STRL33** to function with a wide variety of envelope glycoproteins may become particularly important if therapies are instituted to block other specific co-receptors. We presume that investigations into the roles of **chemokines** and their receptors in lymphocyte biology will provide information important for understanding the pathogenesis of AIDS and for manipulating immune and inflammatory responses for clinical benefit.

L5 ANSWER 20 OF 46 MEDLINE on STN
ACCESSION NUMBER: 1998340702 MEDLINE
DOCUMENT NUMBER: 98340702 PubMed ID: 9676051
TITLE: **Chemokines** and lymphocytes: novel receptors and **HIV**.
AUTHOR: Farber J M
CORPORATE SOURCE: Laboratory of Clinical Investigation, National Institute of Allergy and Infectious Diseases, National Institutes of Health, Bethesda, MD 20892, USA.
SOURCE: JOURNAL OF INVESTIGATIVE MEDICINE, **(1998 Jun)** 46 (5) 197-203. Ref: 32
Journal code: 9501229. ISSN: 1081-5589.
PUB. COUNTRY: United States
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
General Review; (REVIEW)
(REVIEW, TUTORIAL)

LANGUAGE: English
FILE SEGMENT: Priority Journals; AIDS
ENTRY MONTH: 199808
ENTRY DATE: Entered STN: 19980817
Last Updated on STN: 19980817
Entered Medline: 19980806

L5 ANSWER 21 OF 46 MEDLINE on STN
ACCESSION NUMBER: 1998325224 MEDLINE
DOCUMENT NUMBER: 98325224 PubMed ID: 9658152
TITLE: Neutralizing antibodies in sera from macaques immunized with attenuated simian immunodeficiency virus.
AUTHOR: Langlois A J; Desrosiers R C; Lewis M G; KewalRamani V N; Littman D R; Zhou J Y; Manson K; Wyand M S; Bolognesi D P; Montefiori D C
CORPORATE SOURCE: Department of Surgery, Duke University Medical Center, Durham, North Carolina, USA.
CONTRACT NUMBER: AI-35166 (NIAID)
AI28662 (NIAID)
SOURCE: JOURNAL OF VIROLOGY, **(1998 Aug)** 72 (8) 6950-5.
Journal code: 0113724. ISSN: 0022-538X.
PUB. COUNTRY: United States
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals; AIDS
ENTRY MONTH: 199808
ENTRY DATE: Entered STN: 19980817
Last Updated on STN: 19980817
Entered Medline: 19980805

AB Infection with attenuated simian immunodeficiency virus (**SIV**) in rhesus macaques has been shown to raise antibodies capable of neutralizing an animal challenge stock of primary SIVmac251 in CEMx174 cells that correlate with resistance to infection after experimental challenge with this virulent virus (M. S. Wyand, K. H. Manson, M. Garcia-Moll, D. C. Montefiori, and R. C. Desrosiers, J. Virol. 70:3724-3733, 1996). Here we show that these neutralizing antibodies are not detected in human and rhesus peripheral blood mononuclear cells (PBMC). In addition, neutralization of primary SIVmac251 in human and rhesus PBMC was rarely detected with plasma samples from a similar group of animals that had been infected either with SIVmac239Deltanef for 1.5 years or with SIVmac239Delta3 for 3.2 years, although low-level neutralization was detected in CEMx174 cells. Potent neutralization was detected in CEMx174 cells when the latter plasma samples were assessed with laboratory-adapted SIVmac251. In contrast to primary SIVmac251, laboratory-adapted SIVmac251 did not replicate in human and rhesus PBMC despite its ability to utilize CCR5, **Bonzo/STRL33**, and BOB/gpr15 as coreceptors for virus entry. These results illustrate the importance of virus passage history and the choice of indicator cells for making assessments of neutralizing antibodies to lentiviruses such as **SIV**. They also demonstrate that primary SIVmac251 is less sensitive to neutralization in human and rhesus PBMC than it is in established cell lines. Results obtained in PBMC did not support a role for neutralizing antibodies as a mechanism of protection in animals immunized with attenuated **SIV** and challenged with primary SIVmac251.

L5 ANSWER 22 OF 46 MEDLINE on STN
ACCESSION NUMBER: 1998325150 MEDLINE
DOCUMENT NUMBER: 98325150 PubMed ID: 9658078
TITLE: Determinants for sensitivity of human immunodeficiency virus coreceptor CXCR4 to the bicyclam AMD3100.
AUTHOR: Labrosse B; Brelot A; Heveker N; Sol N; Schols D; De Clercq E; Alizon M
CORPORATE SOURCE: INSERM U.332, Institut Cochin de Genetique Moleculaire, 75014 Paris, France.

SOURCE: JOURNAL OF VIROLOGY, **(1998 Aug)** 72 (8) 6381-8.
Journal code: 0113724. ISSN: 0022-538X.
PUB. COUNTRY: United States
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals; AIDS
ENTRY MONTH: 199808
ENTRY DATE: Entered STN: 19980817
Last Updated on STN: 19980817
Entered Medline: 19980805

AB The bicyclam AMD3100 is a potent and selective inhibitor of the replication of human immunodeficiency virus type 1 and type 2 (**HIV**-1 and **HIV**-2). It was recently demonstrated that the compound inhibited **HIV** entry through CXCR4 but not through CCR5. Selectivity of AMD3100 for CXCR4 was further indicated by its lack of effect on **HIV**-1 and **HIV**-2 infection mediated by the CCR5, CCR3, **Bonzo**, BOB, and US28, coreceptors. AMD3100 completely blocked **HIV**-1 infection mediated by a mutant CXCR4 bearing a deletion of most of the amino-terminal extracellular domain. In contrast, relative resistance to AMD3100 was conferred by different single amino acid substitutions in the second extracellular loop (ECL2) or in the adjacent membrane-spanning domain, TM4. Only substitutions of a neutral residue for aspartic acid and of a nonaromatic residue for phenylalanine (Phe) were associated with drug resistance. This suggests a direct interaction of AMD3100 with these amino acids rather than indirect effects of their mutation on the CXCR4 structure. The interaction of aspartic acids of ECL2 and TM4 with AMD3100 is consistent with the positive charge of bicyclams, which might block **HIV**-1 entry by preventing electrostatic interactions between CXCR4 and the **HIV**-1 envelope protein gp120. Other features of AMD3100 must account for its high antiviral activity, in particular the presence of an aromatic linker between the cyclam units. This aromatic group might engage in hydrophobic interactions with the Phe-X-Phe motifs of ECL2 or TM4. These results confirm the importance of ECL2 for the **HIV** coreceptor activity of CXCR4.

L5 ANSWER 23 OF 46 MEDLINE on STN
ACCESSION NUMBER: 1998317009 MEDLINE
DOCUMENT NUMBER: 98317009 PubMed ID: 9653049
TITLE: G protein-coupled receptors in **HIV** and **SIV** entry: new perspectives on lentivirus-host interactions and on the utility of animal models.
AUTHOR: Unutmaz D; KewalRamani V N; Littman D R
CORPORATE SOURCE: Howard Hughes Medical Institute, New York University Medical Center, 540 First Avenue, New York, NY, 10016, USA.
SOURCE: SEMINARS IN IMMUNOLOGY, **(1998 Jun)** 10 (3) 225-36.
Ref: 97
Journal code: 9009458. ISSN: 1044-5323.
PUB. COUNTRY: United States
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
General Review; (REVIEW)
(REVIEW, TUTORIAL)
LANGUAGE: English
FILE SEGMENT: Priority Journals; AIDS
ENTRY MONTH: 199808
ENTRY DATE: Entered STN: 19980820
Last Updated on STN: 20000303
Entered Medline: 19980811

AB Entry of primate lentiviruses into target cells has recently been shown to depend upon the interaction of the viral envelope glycoprotein with CD4 and one or more members of the G protein-coupled receptor (GPCR) family of transmembrane proteins. In vivo, the transmission of **HIV**-1 infection generally requires viral strains that utilise **chemokine** recep- tor CCR5, and these strains prevail during the early course of

infection. Strains isolated later, in the course of progression to immunodeficiency, are often CXCR4-tropic or are dual tropic for both **chemokine** receptors. **SIV** isolates also use CCR5 but are only rarely specific for CXCR4. Instead, **SIVs** use two orphan members of the GPCR family, named **Bonzo/STRL33/ TYMSTR** and BOB/GPR15. Strains of **HIV**-2, which are closely related to the **SIVs**, also often utilise CXCR4, CCR5, BOB and/or **Bonzo**. Additional GPCR family members have also been shown to be utilised by various strains of **HIV** and **SIV** , albeit less efficiently and less frequently. Here we discuss the potential relationship between receptor specificity and viral pathogenesis as well as efforts to develop animal model systems to study the mechanism of disease progression.

L5 ANSWER 24 OF 46 MEDLINE on STN
ACCESSION NUMBER: 1998317008 MEDLINE
DOCUMENT NUMBER: 98317008 PubMed ID: 9653048
TITLE: The function of simian **chemokine** receptors in the replication of **SIV**.
AUTHOR: Marx P A; Chen Z
CORPORATE SOURCE: Aaron Diamond AIDS Research Center, The RockeRfeller University, 455 First Avenue, 7th Floor, New York, NY, 10016, USA.
CONTRACT NUMBER: R01 AI41420-01 (NIAID)
SOURCE: SEMINARS IN IMMUNOLOGY, **(1998 Jun)** 10 (3) 215-23.
Ref: 49
Journal code: 9009458. ISSN: 1044-5323.
PUB. COUNTRY: United States
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
General Review; (REVIEW)
(REVIEW, TUTORIAL)
LANGUAGE: English
FILE SEGMENT: Priority Journals; AIDS
ENTRY MONTH: 199808
ENTRY DATE: Entered STN: 19980820
Last Updated on STN: 19980820
Entered Medline: 19980811

AB The long sought co-receptors for primate lentiviruses were identified as belonging to a large family of cell surface proteins - the seven transmembrane proteins. These proteins normally function as cell surface receptors for **chemokines** and other ligands. The families of genetically divergent Simian Immunodeficiency Viruses (**SIV**), which include the origins of **HIV**-1 and **HIV**-2, use simian and human **chemokine** receptors as their co-receptors. SIVmac, SIVsm, SIVagm and SIVcpz use monkey and human CCR5 for cell fusion and entry. Human-derived **STRL33** (**BONZO**) and human-derived GPR-15 (BOB) are also used, but with variable efficiency. True primary strains of SIVsm, obtained from the naturally infected simian host, the sooty mangabey, use simian and human CCR5 in a strongly CD4 dependent manner. However, some brain and lymphoid isolates from the experimental simian host, the macaque use CCR5 independently of CD4. Unlike T cell line adapted (TCLA) CXCR4-tropic **HIV** strains (XR4 **HIV**), only a few laboratory **SIV** strains use CXCR4 for entry. Macaque and mangabey CXCR4 are fully functional, because they are highly efficient for entry of XR4 **HIV**. The CCR5 co-receptor is used by three of four **SIV** families tested thus far. The fourth family, represented by the isolate, S1Vrcm95GB1, is unique among **SIV** and **HIV** in its use of CCR2b but not CCR5.

L5 ANSWER 25 OF 46 MEDLINE on STN
ACCESSION NUMBER: 1998285765 MEDLINE
DOCUMENT NUMBER: 98285765 PubMed ID: 9621067

TITLE: Exclusive and persistent use of the entry coreceptor CXCR4 by human immunodeficiency virus type 1 from a subject homozygous for CCR5 delta32.
AUTHOR: Michael N L; Nelson J A; KewalRamani V N; Chang G; O'Brien S J; Mascola J R; Volsky B; Louder M; White G C 2nd; Littman D R; Swanstrom R; O'Brien T R
CORPORATE SOURCE: Division of Retrovirology, Walter Reed Army Institute of Research, National Cancer Institute, Rockville, Maryland 20852, USA.
CONTRACT NUMBER: NO1-CP-85649 (NCI)
T32-CA-09156 (NCI)
SOURCE: JOURNAL OF VIROLOGY, **(1998 Jul)** 72 (7) 6040-7.
Journal code: 0113724. ISSN: 0022-538X.
PUB. COUNTRY: United States
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals; AIDS
OTHER SOURCE: GENBANK-AF034375; GENBANK-AF034376; GENBANK-AF034377; GENBANK-AF034378; GENBANK-AF034379; GENBANK-AF034380; GENBANK-AF034381; GENBANK-AF034382; GENBANK-AF034383; GENBANK-AF034384; GENBANK-AF034385
ENTRY MONTH: 199807
ENTRY DATE: Entered STN: 19980713
Last Updated on STN: 20000303
Entered Medline: 19980701

AB Individuals who are homozygous for the 32-bp deletion in the gene coding for the **chemokine** receptor and major human immunodeficiency virus type 1 (**HIV**-1) coreceptor CCR5 (CCR5 -/-) lack functional cell surface CCR5 molecules and are relatively resistant to **HIV** -1 infection. **HIV**-1 infection in CCR5 -/- individuals, although rare, has been increasingly documented. We now report that the viral quasispecies from one such individual throughout disease is homogenous, T cell line tropic, and phenotypically syncytium inducing (SI); exclusively uses CXCR4; and replicates well in CCR5 -/- primary T cells. The recently discovered coreceptors BOB and **Bonzo** are not used. Although early and persistent SI variants have been described in longitudinal studies, this is the first demonstration of exclusive and persistent CXCR4 usage. With the caveat that the earliest viruses available from this subject were from approximately 4 years following primary infection, these data suggest that **HIV**-1 infection can be mediated and persistently maintained by viruses which exclusively utilize CXCR4. The lack of evolution toward the available minor coreceptors in this subject underscores the dominant biological roles of the major coreceptors CCR5 and CXCR4. This and two similar subjects (R. Biti, R. Ffrench, J. Young, B. Bennetts, G. Stewart, and T. Liang, Nat. Med. 3:252-253, 1997; I. Theodoreu, L. Meyer, M. Magierowska, C. Katlama, and C. Rouzioux, Lancet 349:1219-1220, 1997) showed relatively rapid CD4+ T-cell declines despite average or low initial viral RNA load. Since viruses which use CXCR4 exclusively cannot infect macrophages, these data have implications for the relative infection of the T-cell compartment versus the macrophage compartment in vivo and for the development of CCR5-based therapeutics.

L5 ANSWER 26 OF 46 MEDLINE on STN
ACCESSION NUMBER: 1998285695 MEDLINE
DOCUMENT NUMBER: 98285695 PubMed ID: 9620997
TITLE: Genetically divergent strains of human immunodeficiency virus type 2 use multiple coreceptors for viral entry.
AUTHOR: Owen S M; Ellenberger D; Rayfield M; Wiktor S; Michel P; Grieco M H; Gao F; Hahn B H; Lal R B
CORPORATE SOURCE: Retrovirus Diseases Branch, Division of AIDS, STD, and TB Laboratory Research, National Center for Infectious Diseases, Centers for Disease Control and Prevention, Atlanta, Georgia 30333, USA.

CONTRACT NUMBER: AI25291 (NIAID)
AI37466 (NIAID)
SOURCE: JOURNAL OF VIROLOGY, **(1998 Jul)** 72 (7) 5425-32.
Journal code: 0113724. ISSN: 0022-538X.
PUB. COUNTRY: United States
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals; AIDS
ENTRY MONTH: 199807
ENTRY DATE: Entered STN: 19980713
Last Updated on STN: 19980713
Entered Medline: 19980701

AB Several members of the seven-transmembrane **chemokine** receptor family have been shown to serve, with CD4, as coreceptors for entry by human immunodeficiency virus type 1 (**HIV**-1). While coreceptor usage by **HIV**-1 primary isolates has been studied by several groups, there is only limited information available concerning coreceptor usage by primary **HIV**-2 isolates. In this study, we have analyzed coreceptor usage of 15 primary **HIV**-2 isolates, using lymphocytes from a donor with nonfunctional CCR5 (CCR5 -/-; homozygous 32-bp deletion). Based on the infections of PBMCs, seven of these primary isolates had an absolute requirement for CCR5 expression, whereas the remaining eight exhibited a broader coreceptor usage. All CCR5-requiring isolates were non-syncytium inducing, whereas isolates utilizing multiple coreceptors were syncytium inducing. Blocking experiments using known ligands for **chemokine** receptors provided indirect evidence for additional coreceptor utilization by primary **HIV**-2 isolates. Analysis of GHOST4 cell lines expressing various **chemokine** receptors (CCR1, CCR2b, CCR3, CCR4, CCR5, CXCR4, **BONZO**, and BOB) further defined specific coreceptor usage of primary **HIV**-2 isolates. The receptors used included CXCR4, CCR1-5, and the recently described receptors **BONZO** and BOB. However, the efficiency at which the coreceptors were utilized varied greatly among the various isolates. Analysis of V3 envelope sequences revealed no specific motif that correlated with coreceptor usage. Our data demonstrate that primary **HIV**-2 isolates are capable of using a broad range of coreceptors for productive infection in vitro. Additionally, our data suggest that expanded coreceptor usage by **HIV**-2 may correlate with disease progression.

L5 ANSWER 27 OF 46 MEDLINE on STN
ACCESSION NUMBER: 1998070822 MEDLINE
DOCUMENT NUMBER: 98070822 PubMed ID: 9405683
TITLE: CD4-independent, CCR5-dependent infection of brain capillary endothelial cells by a neurovirulent simian immunodeficiency virus strain.
AUTHOR: Edinger A L; Mankowski J L; Doranz B J; Margulies B J; Lee B; Rucker J; Sharron M; Hoffman T L; Berson J F; Zink M C; Hirsch V M; Clements J E; Doms R W
CORPORATE SOURCE: Department of Pathology and Laboratory Medicine, University of Pennsylvania, Philadelphia, PA 19104, USA.
SOURCE: PROCEEDINGS OF THE NATIONAL ACADEMY OF SCIENCES OF THE UNITED STATES OF AMERICA, **(1997 Dec 23)** 94 (26) 14742-7.
Journal code: 7505876. ISSN: 0027-8424.
PUB. COUNTRY: United States
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals; AIDS
ENTRY MONTH: 199802
ENTRY DATE: Entered STN: 19980217
Last Updated on STN: 19980217
Entered Medline: 19980202

AB Brain capillary endothelial cells (BCECs) are targets of CD4-independent

infection by **HIV**-1 and simian immunodeficiency virus (**SIV**) strains in vitro and in vivo. Infection of BCECs may provide a portal of entry for the virus into the central nervous system and could disrupt blood-brain barrier function, contributing to the development of AIDS dementia. We found that rhesus macaque BCECs express **chemokine** receptors involved in **HIV** and **SIV** entry including CCR5, CCR3, CXCR4, and **STRL33**, but not CCR2b, GPR1, or GPR15. Infection of BCECs by the neurovirulent strain **SIV**/17E-Fr was completely inhibited by aminooxypentane regulation upon activation, normal T cell expression and secretion in the presence or absence of ligands, but not by eotaxin or antibodies to CD4. We found that the envelope (env) proteins from **SIV**/17E-Fr and several additional **SIV** strains mediated cell-cell fusion and virus infection with CD4-negative, CCR5-positive cells. In contrast, fusion with cells expressing the coreceptors **STRL33**, GPR1, and GPR15 was CD4-dependent. These results show that CCR5 can serve as a primary receptor for **SIV** in BCECs and suggest a possible CD4-independent mechanism for blood-brain barrier disruption and viral entry into the central nervous system.

L5 ANSWER 28 OF 46 MEDLINE on STN
ACCESSION NUMBER: 1998022660 MEDLINE
DOCUMENT NUMBER: 98022660 PubMed ID: 9359702
TITLE: In vivo evolution of **HIV**-1 co-receptor usage and sensitivity to **chemokine**-mediated suppression.
AUTHOR: Scarlatti G; Tresoldi E; Bjorndal A; Fredriksson R; Colognesi C; Deng H K; Malnati M S; Plebani A; Siccardi A G; Littman D R; Fenyo E M; Lusso P
CORPORATE SOURCE: Unit of Immunobiology of HIV, DIBIT, San Raffaele Scientific Institute, Milan, Italy.
SOURCE: NATURE MEDICINE, **(1997 Nov)** 3 (11) 1259-65.
Journal code: 9502015. ISSN: 1078-8956.
PUB. COUNTRY: United States
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals; AIDS
OTHER SOURCE: GENBANK-AF023305; GENBANK-AF023306; GENBANK-AF023307; GENBANK-AF023308; GENBANK-AF023309; GENBANK-AF023310; GENBANK-AF023311; GENBANK-AF023312; GENBANK-AF023313; GENBANK-AF023314; GENBANK-AF023315; GENBANK-AF023316; GENBANK-AF023317; GENBANK-AF023318; GENBANK-AF023319; GENBANK-AF023320; GENBANK-AF023321; GENBANK-AF023322; GENBANK-AF023323; GENBANK-AF023324; GENBANK-AF023325; GENBANK-AF023326; GENBANK-AF023327; GENBANK-AF023328; GENBANK-AF023329; GENBANK-AF023330; GENBANK-AF023331; GENBANK-AF023332; GENBANK-AF023333; GENBANK-AF023334; +
ENTRY MONTH: 199712
ENTRY DATE: Entered STN: 19980109
Last Updated on STN: 19980109
Entered Medline: 19971209

AB Following the identification of the C-C **chemokines** RANTES, MIP-1alpha and MIP-1beta as major human immunodeficiency virus (**HIV**)-suppressive factors produced by CD8+ T cells, several **chemokine** receptors were found to serve as membrane co-receptors for primate immunodeficiency lentiretroviruses. The two most widely used co-receptors thus far recognized, CCR5 and CXCR4, are expressed by both activated T lymphocytes and mononuclear phagocytes. CCR5, a specific RANTES, MIP-1alpha and MIP-1 receptor, is used preferentially by non-MT2-tropic **HIV**-1 and **HIV**-2 strains and by simian immunodeficiency virus (**SIV**), whereas CXCR4, a receptor for the C-X-C **chemokine** SDF-1, is used by MT2-tropic **HIV**-1 and **HIV**-2, but not by **SIV**. Other receptors with a more restricted cellular distribution, such as CCR2b, CCR3 and **STRL33** , can also function as co-receptors for selected viral isolates. The

third variable region (V3) of the gp120 envelope glycoprotein of **HIV**-1 has been fingered as a critical determinant of the co-receptor choice. Here, we document a consistent pattern of evolution of viral co-receptor usage and sensitivity to **chemokine**-mediated suppression in a longitudinal follow-up of children with progressive **HIV**-1 infection. Viral isolates obtained during the asymptomatic stages generally used only CCR5 as a co-receptor and were inhibited by RANTES, MIP-1alpha and MIP-1beta, but not by SDF-1. By contrast, the majority of the isolates derived after the progression of the disease were resistant to C-C **chemokines**, having acquired the ability to use CXCR4 and, in some cases, CCR3, while gradually losing CCR5 usage. Surprisingly, most of these isolates were also insensitive to SDF-1, even when used in combination with RANTES. An early acquisition of CXCR4 usage predicted a poor prognosis. In children who progressed to AIDS without a shift to CXCR4 usage, all the sequential isolates were CCR5-dependent but showed a reduced sensitivity to C-C **chemokines**. Discrete changes in the V3 domain of gp120 were associated with the loss of sensitivity to C-C **chemokines** and the shift in co-receptor usage. These results suggest an adaptive evolution of **HIV**-1 in vivo, leading to escape from the control of the antiviral C-C **chemokines**.

L5 ANSWER 29 OF 46 MEDLINE on STN
ACCESSION NUMBER: 97431687 MEDLINE
DOCUMENT NUMBER: 97431687 PubMed ID: 9285716
TITLE: **TYMSTR**, a putative **chemokine** receptor selectively expressed in activated T cells, exhibits **HIV**-1 coreceptor function.
AUTHOR: Loetscher M; Amara A; Oberlin E; Brass N; Legler D; Loetscher P; D'Apuzzo M; Meese E; Rousset D; Virelizier J L; Baggiolini M; Arenzana-Seisdedos F; Moser B
CORPORATE SOURCE: Theodor-Kocher Institute University of Bern P.O. Box 99, CH-3000 Bern 9, Switzerland.
SOURCE: CURRENT BIOLOGY, **(1997 Sep 1)** 7 (9) 652-60. Journal code: 9107782. ISSN: 0960-9822.
PUB. COUNTRY: ENGLAND: United Kingdom
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals; AIDS
ENTRY MONTH: 199802
ENTRY DATE: Entered STN: 19980217
Last Updated on STN: 19980217
Entered Medline: 19980203

AB BACKGROUND: **Chemokines** bind to specific receptors and mediate leukocyte migration to sites of inflammation. Recently, some **chemokine** receptors, notably CXCR4 and CCR5, have been shown to be essential fusion factors on target cells for infection by human immunodeficiency virus (**HIV**); the **chemokines** bound by these receptors have also been shown to act as potent inhibitors of **HIV** infection. Here, we describe the isolation of a novel, putative **chemokine** receptor. RESULTS: We have isolated the cDNA for a putative human **chemokine** receptor, which we have termed **TYMSTR** (T-lymphocyte-expressed seven-transmembrane domain receptor). The **TYMSTR** gene is localized to human chromosome 3 and encodes a protein that has a high level of identity with **chemokine** receptors. **TYMSTR** mRNA was selectively expressed in interleukin-2-stimulated T lymphocytes but not in freshly isolated lymphocytes and leukocytes or related cell lines. The natural ligand for **TYMSTR** was not identified among 32 human **chemokines** and other potential ligands. Cells co-expressing **TYMSTR** and human CD4 fused with cells expressing envelope glycoproteins of macrophage (M)-tropic **HIV**-1 as well as T-cell line (T)-tropic **HIV**-1 isolates. Addition of infectious, T-tropic **HIV**-1 particles to **TYMSTR**/CD4-expressing

cells resulted in viral entry and proviral DNA formation. CONCLUSIONS: Our findings demonstrate that **TYMSTR**, in combination with CD4, mediates **HIV**-1 fusion and entry. The high-level expression of **TYMSTR** in CD4(+) T lymphocytes and the selectivity of this receptor for T-tropic and M-tropic **HIV**-1 strains indicates that **TYMSTR** might function as **HIV** coreceptor at both early and late stages of infection.

L5 ANSWER 30 OF 46 MEDLINE on STN
ACCESSION NUMBER: 97373948 MEDLINE
DOCUMENT NUMBER: 97373948 PubMed ID: 9230431
TITLE: A new **SIV** co-receptor, **STRL33**.
COMMENT: Comment in: Nature. 1997 Jul 17;388(6639):230-1
AUTHOR: Alkhatib G; Liao F; Berger E A; Farber J M; Peden K W
SOURCE: NATURE, **(1997 Jul 17)** 388 (6639) 238.
Journal code: 0410462. ISSN: 0028-0836.
PUB. COUNTRY: ENGLAND: United Kingdom
DOCUMENT TYPE: Letter
LANGUAGE: English
FILE SEGMENT: Priority Journals; AIDS
ENTRY MONTH: 199708
ENTRY DATE: Entered STN: 19970813
Last Updated on STN: 19970813
Entered Medline: 19970807

L5 ANSWER 31 OF 46 MEDLINE on STN
ACCESSION NUMBER: 97373944 MEDLINE
DOCUMENT NUMBER: 97373944 PubMed ID: 9230427
TITLE: Immunodeficiency viruses. Spoilt for choice of co-receptors.
COMMENT: Comment on: Nature. 1997 Jul 17;388(6639):238
Comment on: Nature. 1997 Jul 17;388(6639):296-300
AUTHOR: Clapham P R; Weiss R A
SOURCE: NATURE, **(1997 Jul 17)** 388 (6639) 230-1.
Journal code: 0410462. ISSN: 0028-0836.
PUB. COUNTRY: ENGLAND: United Kingdom
DOCUMENT TYPE: Commentary
News Announcement
LANGUAGE: English
FILE SEGMENT: Priority Journals; AIDS
ENTRY MONTH: 199708
ENTRY DATE: Entered STN: 19970813
Last Updated on STN: 19970813
Entered Medline: 19970807

L5 ANSWER 32 OF 46 MEDLINE on STN
ACCESSION NUMBER: 97311099 MEDLINE
DOCUMENT NUMBER: 97311099 PubMed ID: 9166430
TITLE: **STRL33**, A novel **chemokine** receptor-like protein, functions as a fusion cofactor for both macrophage-tropic and T cell line-tropic **HIV**-1.
AUTHOR: Liao F; Alkhatib G; Peden K W; Sharma G; Berger E A; Farber J M
CORPORATE SOURCE: Laboratory of Clinical Investigation, National Institute of Allergy and Infectious Diseases, National Institutes of Health, Bethesda, Maryland, USA.
SOURCE: JOURNAL OF EXPERIMENTAL MEDICINE, **(1997 Jun 2)** 185 (11) 2015-23.
Journal code: 2985109R. ISSN: 0022-1007.
PUB. COUNTRY: United States
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals; AIDS
OTHER SOURCE: GENBANK-U73529; GENBANK-U73530; GENBANK-U73531

ENTRY MONTH: 199706
ENTRY DATE: Entered STN: 19970716
Last Updated on STN: 20000303
Entered Medline: 19970630

AB The **chemokine** receptors CXCR4, CCR2B, CCR3, and CCR5 have recently been shown to serve along with CD4 as coreceptors for **HIV**-1. The tropisms of **HIV**-1 strains for subgroups of CD4(+) cells can be explained, at least partly, by the selective use of G protein-coupled receptors (GPCRs). We have identified a novel human gene, **STRL33**, located on chromosome 3 that encodes a GPCR with sequence similarity to **chemokine** receptors and to **chemokine** receptor-like orphan receptors. **STRL33** is expressed in lymphoid tissues and activated T cells, and is induced in activated peripheral blood lymphocytes. When transfected into nonhuman NIH 3T3 cells expressing human CD4, the **STRL33** cDNA rendered these cells competent to fuse with cells expressing **HIV**-1 envelope glycoproteins (Envs). Of greatest interest, **STRL33**, in contrast with CXCR4 or CCR5, was able to function as a cofactor for fusion mediated by Envs from both T cell line-tropic and macrophage-tropic **HIV**-1 strains. **STRL33**-transfected Jurkat cell lines also supported enhanced productive infection with **HIV**-1 compared with control Jurkat cells. Despite the sequence similarities between **STRL33** and **chemokine** receptors, **STRL33**-transfected cell lines did not respond to any in a panel of **chemokines**. Based on the pattern of tissue expression of the **STRL33** mRNA, and given the ability of **STRL33** to function with Envs of differing tropisms, **STRL33** may play a role in the establishment and/or progression of **HIV**-1 infection.

L5 ANSWER 33 OF 46 CAPLUS COPYRIGHT 2003 ACS on STN
ACCESSION NUMBER: 2003:199452 CAPLUS
DOCUMENT NUMBER: 138:198582
TITLE: **Chemokine** receptor CCR-interacting MIP-1.alpha. peptide and its use in treatment of **HIV** infections
INVENTOR(S): Albini, Adriana; Noonan, Douglas; Benelli, Roberto; Giunciuglio, Daniela
PATENT ASSIGNEE(S): Istituto Nazionale per la Ricerca sul Cancro, Italy
SOURCE: Ital. Appl., 18 pp.
CODEN: ITXXCZ
DOCUMENT TYPE: Patent
LANGUAGE: Italian
FAMILY ACC. NUM. COUNT: 1
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
IT 98MI1189	A1	19991129	IT 1998-MI1189	19980529 <--

PRIORITY APPLN. INFO.: IT 1998-MI1189 19980529
AB The title MIP-1.alpha. peptides, esp. PTACCFSYTSRQIPQNFIADYFETSS (I), which bind to **chemokine** receptors CCR, can be used to treat **HIV** infections. Thus, I was found to be a chemoattractant for monocytes and to stimulate Ca2+ transport in these cells. I inhibited **HIV**-1 and **HIV**-2 infection mediated by CXCR4, CCR5, and CCR3 as well as CCR-2b, BOB, **BONZO**, and V-28.

L5 ANSWER 34 OF 46 CAPLUS COPYRIGHT 2003 ACS on STN
ACCESSION NUMBER: 1999:641083 CAPLUS
DOCUMENT NUMBER: 131:281536
TITLE: Orphan receptor HBMBU14 and PF-4 for PF-4 receptor agonist and antagonist assays
INVENTOR(S): Macphee, Colin Houston; Moores, Kitty; Berkhout, Theodorus Antonius
PATENT ASSIGNEE(S): Smithkline Beecham Plc, UK

SOURCE: PCT Int. Appl., 35 pp.
CODEN: PIXXD2
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 1
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE	
WO 9950670	A1	19991007	WO 1999-GB950	19990326	<--
W: JP					
RW: AT, BE, CH, CY, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE					
US 6232084	B1	20010515	US 1999-275384	19990324	
EP 1066526	A1	20010110	EP 1999-913452	19990326	
R: BE, CH, DE, DK, FR, GB, IT, LI, NL					
JP 2002510053	T2	20020402	JP 2000-541527	19990326	

PRIORITY APPLN. INFO.: GB 1998-6677 A 19980327
WO 1999-GB950 W 19990326

AB The ligand PF-4 has been identified as a ligand for the 7TM orphan receptor HBMBU14, also known as **TYMSTR**, STRL-33 and **BONZO**.

REFERENCE COUNT: 2 THERE ARE 2 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L5 ANSWER 35 OF 46 CAPLUS COPYRIGHT 2003 ACS on STN
ACCESSION NUMBER: 1999:359675 CAPLUS
DOCUMENT NUMBER: 131:13989
TITLE: Virus vectors expressing genes for ligands of CD4-associated **chemokine** receptors for inhibition or delay of the binding of an immunodeficiency virus to cells
INVENTOR(S): Mehtali, Majid; Sorg, Tania; Calenda, Valerie; Marigliano, Martine
PATENT ASSIGNEE(S): Transgene S.A., Fr.
SOURCE: PCT Int. Appl., 46 pp.
CODEN: PIXXD2
DOCUMENT TYPE: Patent
LANGUAGE: French
FAMILY ACC. NUM. COUNT: 1
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE	
WO 9927122	A1	19990603	WO 1998-FR2503	19981123	<--
W: AU, CA, JP, US					
RW: AT, BE, CH, CY, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE					
FR 2771423	A1	19990528	FR 1997-14672	19971121	<--
AU 9912463	A1	19990615	AU 1999-12463	19981123	<--

PRIORITY APPLN. INFO.: FR 1997-14672 19971121
WO 1998-FR2503 19981123

AB A method of preventing or delaying immunodeficiency virus entry into cells using viruses expressing genes for ligands of CD4-assocd. **chemokine** receptors is described. Preferably, the virus is an attenuated adenovirus lacking a no. early genes and the ligand may be any of several **chemokines** including RANTES, macrophage-derived **chemokine**, MIP-1.alpha. or MIP-1.beta., or stromal cell-derived factor.

REFERENCE COUNT: 21 THERE ARE 21 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L5 ANSWER 36 OF 46 CAPLUS COPYRIGHT 2003 ACS on STN
ACCESSION NUMBER: 1999:77594 CAPLUS

DOCUMENT NUMBER: 130:152567
TITLE: Expression cloning of alternate receptors **Bonzo** and BOB used for cell entry by simian and human immunodeficiency viruses
INVENTOR(S): Littman, Dan R.; Deng, Hongkui; Unutmaz, Derya; Kewalramani, Vineet N.
PATENT ASSIGNEE(S): New York University, USA
SOURCE: PCT Int. Appl., 97 pp.
CODEN: PIXXD2
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 2
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 9903888	A1	19990128	WO 1998-US14857	19980717 <--

W: CA, IL, JP
RW: AT, BE, CH, CY, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE

PRIORITY APPLN. INFO.: US 1997-896155 A 19970717

AB The present invention provides two new **HIV/SIV** translocation promoting agents, **Bonzo** and BOB. An expression-cloning strategy was used to identify **SIV** receptors and isolate genes encoding 2 members of the seven-transmembrane G-protein-coupled receptor family that are used not only by **SIVs**, but also by strains of **HIV**-2 and M-tropic **HIV**-1. The present invention also provides the amino acid and DNA sequences of human, African green monkey, and pigtail macaque of the receptor proteins **Bonzo** and BOB. Both receptors are closely related to the **chemokine**-receptor family and are expressed in lymphoid tissues. Mammalian cells transfected with **Bonzo** and/or BOB and human CD4 as well as antibodies to the receptor **Bonzo** are also included. Furthermore, a method of identifying other such translocation promoting agents is also disclosed. Diagnostic and therapeutic uses of the translocation promoting agents of the present invention are also provided. Usage of these new receptors following exptl. infection of non-human primates with **SIV** strains may provide important insight into viral transmission and the mechanisms of **SIV**- and **HIV**-induced acquired immune-deficiency syndrome.

REFERENCE COUNT: 2 THERE ARE 2 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L5 ANSWER 37 OF 46 CAPLUS COPYRIGHT 2003 ACS on STN
ACCESSION NUMBER: 1998:684950 CAPLUS
DOCUMENT NUMBER: 129:271564
TITLE: **STRL33**, a human fusion accessory factor associated with **hiv** infection
INVENTOR(S): Farber, Joshua M.; Liao, Fang; Alkhatib, Ghalib; Berger, Edward A.
PATENT ASSIGNEE(S): United States Dept. of Health and Human Services, USA
SOURCE: PCT Int. Appl., 75 pp.
CODEN: PIXXD2
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 1
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 9844098	A2	19981008	WO 1998-US6517	19980331 <--
WO 9844098	A3	19981223		

W: AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, CA, CH, CN, CU, CZ, DE, DK, EE, ES, FI, GB, GE, GH, GM, GW, HU, ID, IL, IS, JP, KE, KG,

KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MD, MG, MK, MN, MW, MX,
NO, NZ, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TR, TT,
UA, UG, US, UZ, VN, YU, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM
RW: GH, GM, KE, LS, MW, SD, SZ, UG, ZW, AT, BE, CH, DE, DK, ES, FI,
FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, BF, BJ, CF, CG, CI, CM,
GA, GN, ML, MR, NE, SN, TD, TG

AU 9867946 A1 19981022 AU 1998-67946 19980331 <--
EP 979272 A1 20000216 EP 1998-913379 19980331
R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT,
IE, FI

PRIORITY APPLN. INFO.: US 1997-42880P P 19970331
WO 1998-US6517 W 19980331

AB The susceptibility to human immunodeficiency virus (**HIV**) infection depends on the cell surface expression of the human CD4 mol. and a human fusion accessory factor assocd. with **HIV** infection (**STRL33**). **STRL33** is a member of the 7-transmembrane segment superfamily of G-protein-coupled cell surface mols. **STRL33** plays a role in the membrane fusion step of **HIV** infection for both TCL-tropic and M-tropic variants of **HIV**. The invention provides **STRL33** polypeptide and polynucleotide sequences encoding **STRL33** polypeptide. The establishment of stable, nonhuman cell lines and transgenic mammals having cells that coexpress human CD4 and **STRL33** provides valuable tools for the continuing research of **HIV** infection and the development of more effective anti-**HIV** therapeutics. In addn., antibodies against **STRL33**, isolated and purified peptide fragments of **STRL33** , and **STRL33**-binding biol. agents, capable of blocking membrane fusion between **HIV** target cells represent potential anti-**HIV** therapeutics.

L5 ANSWER 38 OF 46 CAPLUS COPYRIGHT 2003 ACS on STN
ACCESSION NUMBER: 1998:540411 CAPLUS
DOCUMENT NUMBER: 129:244026
TITLE: Neutralization profiles of primary human immunodeficiency virus type 1 isolates in the context of coreceptor usage
AUTHOR(S): Cecilia, D.; Kewalramani, Vineet N.; O'leary, Jeanne; Volsky, Barbara; Nyambi, Phillipe; Burda, Sherri; Xu, Serena; Littman, Dan R.; Zolla-Pazner, Susan
CORPORATE SOURCE: New York University Medical Center, New York, NY, USA
SOURCE: Journal of Virology (**1998**), 72(9), 6988-6996
CODEN: JOVIAM; ISSN: 0022-538X
PUBLISHER: American Society for Microbiology
DOCUMENT TYPE: Journal
LANGUAGE: English

AB Most strains of human immunodeficiency virus type 1 (**HIV**-1) which have only been carried in vitro in peripheral blood mononuclear cells (primary isolates) can be neutralized by antibodies, but their sensitivity to neutralization varies considerably. To study the parameters that contribute to the differential neutralization sensitivity of primary **HIV**-1 isolates, the authors developed a neutralization assay with a panel of genetically engineered cell lines (GHOST cells) that express CD4, one of eight **chemokine** receptors which function as **HIV**-1 coreceptors, and a Tat-dependent green fluorescent protein reporter cassette which permits the evaluation and quantitation of **HIV**-1 infection by flow cytometry. All 21 primary isolates from several clades could grow in the various GHOST cell lines, and their use of one or more coreceptors could easily be defined by flow cytometric anal. Ten of these primary isolates, three that were CXCR4 (X4)-tropic, three that were CCR5-tropic, and four that were dual- or polytropic were chosen for study of their sensitivity to neutralization by human monoclonal and polyclonal antibodies. Viruses from the X4-tropic category of viruses were first tested since they have generally been considered to be particularly neutralization sensitive. It was found that

the X4-tropic virus group contained both neutralization-sensitive and neutralization-resistant viruses. Similar results were obtained with R5-tropic viruses and with dual- or polytropic viruses. Within each category of viruses, neutralization sensitivity and resistance could be obsd. Therefore, sensitivity to neutralization appears to be the consequence of factors that influence the antibody-virus interaction and its sequelae rather than coreceptor usage. Neutralization of various viruses by the V3-specific monoclonal antibody, 447-52D, was shown to be dependent not only on the presence of the relevant epitope but also on its presentation. An epitope within the envelope of a particular virus is not sufficient to render a virus sensitive to neutralization by an antibody that recognizes that epitope. Moreover, conformation-dependent factors may overcome the need for abs. fidelity in the match between an antibody and its core epitope, permitting sufficient affinity between the viral envelope protein and the antibody to neutralize the virus. The studies indicate that the neutralization sensitivity of **HIV**-1 primary isolates is a consequence of the complex interaction between virus, antibody, and target cell.

REFERENCE COUNT: 54 THERE ARE 54 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L5 ANSWER 39 OF 46 CAPLUS COPYRIGHT 2003 ACS on STN

ACCESSION NUMBER: 1997:470456 CAPLUS
DOCUMENT NUMBER: 127:219358
TITLE: Expression cloning of new receptors used by simian and human immunodeficiency viruses
AUTHOR(S): Deng, HongKui; Unutmaz, Derya; KewalRamani, Vineet N.; Littman, Dan R.
CORPORATE SOURCE: Div. Mol. Pathogen.,Skirball Inst. Biomol. Med., Howard Huthes Med. Inst., New York Univ. Med. Cent., New York, NY, 10016, USA
SOURCE: Nature (London) (**1997**), 388(6639), 296-300
CODEN: NATUAS; ISSN: 0028-0836
PUBLISHER: Macmillan Magazines
DOCUMENT TYPE: Journal
LANGUAGE: English

AB Several members of the **chemokine**-receptor family serve, in conjunction with CD4, as receptors for the entry of human immunodeficiency virus type I (**HIV**-1) into cells. The principal receptor for entry of macrophage-tropic (M-tropic) **HIV**-1 strains in CCR5, whereas that for T-cell-line-tropic (T-tropic) strains is CXR4. Unlike **HIV**-1, infection with either M-tropic or T-tropic strains of simian immunodeficiency virus (**SIV**) can be mediated by CCR5, but not CXCR4 (refs 7-10). **SIV** strains will also infect CD4+ cells that lack CCR5, which suggests that these strains use as yet unidentified receptors. Here the authors use an expression-cloning strategy to identify **SIV** receptors and have isolated genes encoding two members of the seven-transmembrane G-protein-coupled receptor family that are used not only by **SIVs**, but also by strains of **HIV** -2 and M-tropic **HIV**-1. Both receptors are closely related to the **chemokine**-receptor family and are expressed in lymphoid tissues. One of the receptors is also expressed in colon and may therefore be important in viral transmission. Usage of these new receptors following exptl. infection of non-human primates with **SIV** strains may provide important insight into viral transmission and the mechanisms of **SIV**- and **HIV**-induced acquired immune-deficiency syndrome.

L5 ANSWER 40 OF 46 CAPLUS COPYRIGHT 2003 ACS on STN

ACCESSION NUMBER: 1997:470220 CAPLUS
DOCUMENT NUMBER: 127:202585
TITLE: A new **SIV** co-receptor, **STRL33**
AUTHOR(S): Alkhatib, Ghalib; Liao, Fang; Berger, Edward A.; Farber, Joshua M.; Peden Keith W. C.

CORPORATE SOURCE: Lab. Viral Dis., NIAID, Natl. Inst. Health, Bethesda, MD, 20892, USA
SOURCE: Nature (London) (**1997**), 388(6639), 238
CODEN: NATUAS; ISSN: 0028-0836
PUBLISHER: Macmillan Magazines
DOCUMENT TYPE: Journal
LANGUAGE: English

AB The authors report here that **STRL33**, a **chemokine** receptor-like orphan receptor expressed in activated human lymphocytes and acting as a fusion co-factor with envelope glycoproteins (Envs) from **HIV**-1 strains of various tropisms, is a co-receptor for **SIV**. These findings demonstrate that **STRL33** is active with a broader range of Envs than has been discovered for any of the co-receptors so far discovered. **STRL33** will thus be of particular value in unraveling the structural determinants of interactions between Envs and co-receptors.

L5 ANSWER 41 OF 46 EMBASE COPYRIGHT 2003 ELSEVIER SCI. B.V. on STN
ACCESSION NUMBER: 97226930 EMBASE
DOCUMENT NUMBER: 1997226930
TITLE: A new **SIV** co-receptor, **STRL33** [8].
AUTHOR: Alkhatib G.; Liao F.; Berger E.A.; Farber J.M.; Peden K.W.C.
CORPORATE SOURCE: G. Alkhatib, Laboratory of Viral Diseases, NIADID, National Institutes of Health, Bethesda, MD 20892, United States
SOURCE: Nature, (1997) 388/6639 (238).
Refs: 18
ISSN: 0028-0836 CODEN: NATUAS
COUNTRY: United Kingdom
DOCUMENT TYPE: Journal; Letter
FILE SEGMENT: 004 Microbiology
026 Immunology, Serology and Transplantation
LANGUAGE: English

L5 ANSWER 42 OF 46 BIOSIS COPYRIGHT 2003 BIOLOGICAL ABSTRACTS INC. on STN
ACCESSION NUMBER: 2000:99826 BIOSIS
DOCUMENT NUMBER: PREV200000099826
TITLE: **Chemokines** and chemokin receptor on simian astrocytes.
AUTHOR(S): Croitoru, J. (1); Guillemin, G.; Boussin, F. D.; Mognetti, B.; Lebel-Binay, S.; Leveque, T.; Gras, G.; Le Grand, R.; Dormont, D.
CORPORATE SOURCE: (1) C.R.S.S.A., La Tronche, Grenoble France
SOURCE: Travaux Scientifiques des Chercheurs du Service de Sante des Armees, (1999) Vol. 0, No. 20, pp. 141-142.
ISSN: 0243-7473.
DOCUMENT TYPE: Article
LANGUAGE: French
SUMMARY LANGUAGE: English; French

AB In this study, we have investigated **chemokine** production by adult simian astrocytes, that occurs after stimulation with pro-inflammatory cytokines. Detection of **chemokines** and their receptor mRNAs was performed using specific RT-PCR amplifications after in vitro stimulation with TNF-alpha and IFN-gamma. Proteins were detected by ELISA techniques. Among the **chemokines** tested on 3 different cultures of adult simian astrocytes, we found an enhanced expression of RANTES, IP-10, MCP-1 and HuMIG mRNA 72 hours after stimulation with TNF-alpha. In addition, pretreatment with IFN-gamma significantly increased production of these **chemokines**. Furthermore, we have detected the expression of mRNA encoding for receptors CXCR4 (LESTR/fusin), GPR1, BOB (GPR15) and **Bonzo** (**STRL33**), cofactors for fusion and entry of **HIV**-1 and **SIV**.

L5 ANSWER 43 OF 46 BIOSIS COPYRIGHT 2003 BIOLOGICAL ABSTRACTS INC. on STN

ACCESSION NUMBER: 1999:368953 BIOSIS
DOCUMENT NUMBER: PREV199900368953
TITLE: Blocking **HIV** co-receptors by **chemokines**
.
AUTHOR(S): Virelizier, J. L. (1)
CORPORATE SOURCE: (1) Unite d'Immunologie Virale, Institut Pasteur, 28, rue du Dr Roux, F-75724, Paris Cedex 15 France
SOURCE: Brown, F. [Editor]; Mire-Sluis, T. [Editor]. Developments in Biological Standardization, (1999) Vol. 97, pp. 105-109. Developments in Biological Standardization; Biological characterization and assay of cytokines and growth factors. Publisher: S. Karger AG P.O. Box, Allschwilerstrasse 10, CH-4009 Basel, Switzerland.
Meeting Info.: Meeting held at the National Institute for Biological Standards and Control Herts, England, UK September 10-12, 1997
ISSN: 0301-5149. ISBN: 3-8055-6895-9.
DOCUMENT TYPE: Book; Conference
LANGUAGE: English

L5 ANSWER 44 OF 46 BIOSIS COPYRIGHT 2003 BIOLOGICAL ABSTRACTS INC. on STN
ACCESSION NUMBER: 1999:99153 BIOSIS
DOCUMENT NUMBER: PREV199900099153
TITLE: Quantification of CD4, CCR5, CXCR4, and **STRL33** levels on differentially conditioned monocyte-derived macrophages, various subsets of peripheral blood leukocytes and CD34+ bone marrow progenitor cells.
AUTHOR(S): Lee, B. (1); Sharron, M.; Tsang, M.; Majka, M. M.; Ratajczak, M. Z.; Montaner, L. J.; Weissman, D.; Doms, R. W.
CORPORATE SOURCE: (1) Wistar Inst., Philadelphia, PA USA
SOURCE: Blood, (**Nov. 15, 1998**) Vol. 92, No. 10 SUPPL. 1 PART 1-2, pp. 164A.
Meeting Info.: 40th Annual Meeting of the American Society of Hematology Miami Beach, Florida, USA December 4-8, 1998 The American Society of Heamatology
. ISSN: 0006-4971.
DOCUMENT TYPE: Conference
LANGUAGE: English

L5 ANSWER 45 OF 46 BIOSIS COPYRIGHT 2003 BIOLOGICAL ABSTRACTS INC. on STN
ACCESSION NUMBER: 1999:99103 BIOSIS
DOCUMENT NUMBER: PREV199900099103
TITLE: Biological consequences of **chemokine** receptor polarization on TH1 and TH2 cells from individuals wildtype for CCR5 or homozygous for the DELTA32 CCR5 allele.
AUTHOR(S): Lee, B. (1); Bailer, R. T.; Rucker, J.; Tsang, M.; Doms, R. W.; Montaner, L. J.
CORPORATE SOURCE: (1) Wistar Inst., Philadelphia, PA USA
SOURCE: Blood, (**Nov. 15, 1998**) Vol. 92, No. 10 SUPPL. 1 PART 1-2, pp. 21A.
Meeting Info.: 40th Annual Meeting of the American Society of Hematology Miami Beach, Florida, USA December 4-8, 1998 The American Society of Heamatology
. ISSN: 0006-4971.
DOCUMENT TYPE: Conference
LANGUAGE: English

L5 ANSWER 46 OF 46 BIOSIS COPYRIGHT 2003 BIOLOGICAL ABSTRACTS INC. on STN
ACCESSION NUMBER: 1998:21451 BIOSIS
DOCUMENT NUMBER: PREV199800021451
TITLE: **Chemokine** receptors and animal models for **HIV** pathogenesis.
AUTHOR(S): Littman, Dan R.; Davis, Craig; Deng, Hongkui; Ellmeier,

Wilfried; Hill, Mark; Kewalramani, Vineet; Scarborough, John; Taniuchi, Ichiro; Unutmaz, Derya; Zou, Yongrui
CORPORATE SOURCE: Howard Hughes Med. Inst., Skirball Inst. Biomolecular Med., NYU Med. Cent., New York, NY, 10016 USA
SOURCE: Molecular Biology of the Cell, (**Nov., 1997**) Vol. 8, No. SUPPL., pp. 349A.
Meeting Info.: 37th Annual Meeting of the American Society for Cell Biology Washington, D.C., USA December 13-17, 1997 American Society for Cell Biology
. ISSN: 1059-1524.
DOCUMENT TYPE: Conference
LANGUAGE: English